A Proposed Causal Model For 'Near-Miss Effect' in Gambling Disorder

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Abstract

Gambling disorder, according to DSM-V, has an essential feature of persistent and recurrent maladaptive gambling behavior that disrupts personal, family, and/or vocational pursuits. Cognitive distortions can give rise to abnormal responses to specific events related to gambling, which in this case is the near-miss loss, typically present in slot machines as two jackpot symbols lining up perfectly, but the player loses on the third one, leading to frustration but motivation to continue playing. We have came up with a possible causal model of these brain areas, namely Insula, Ventral Striatum, Superior Colliculus and Substantia Nigra, connections that processes these near-miss loss results as an 'almost win' situation. Results showed that our model has demonstrated possible causal connections explaining neuronal activities in brain areas of an individual in different states, high and low gambling severity, going through different gambling outcomes, wins, full-miss losses and near-miss losses. Further research to optimize the model based on the present limitations and possibility for an extension of the model and also its real world applications are discussed.

1 Introduction

While gambling is prevalent and enjoyed recreationally by a vast majority, in a significant minority, it leads to the development of a gambling disorder which leads to much distress and problems for the individuals and families. DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, the fifth version) classifies gambling disorder under the category of 'Substance-Related and Addictive Disorders' (American Psychiatric Association, 2013). Many similarities have been observed between substance use disorders and gambling disorder (Lindberg, Clark, & Bowden-Jones, 2014).

There is a widespread notion among recreational gamblers that 'the house always wins' – the cumulative expected value of gambling is negative overtime and often leads to loss of money for the player (Clark, Lawrence, Astley-Jones, & Gray, 2009). As such, in pathological gamblers, the persistence of the problem and their addiction to gambling despite the negative outcomes is suggested to be due to distorted beliefs about the probability of success in the future outcomes (Joukhador, Maccallum, & Blaszczynski, 2003). This biased processing of chance, probability and skill in pathological gamblers is known as cognitive distortion.

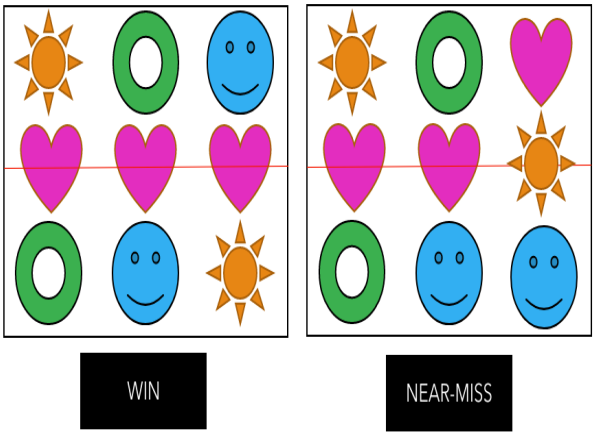

Figure 1: Win and Near-Miss Loss Outcomes in Three-Reel Slot Machine

There are a multitude of cognitive distortions experienced by pathological gamblers including Losses Disguised as Wins (LDWs) and Near-Misses (NMs) which are common in slot machines. LDWs, defined as scenarios such that a player wins less money than they bet, resulting in an overall loss, typically occur in multiline slot machines, which player has a chance to win small amount of money in various combinations of the symbols present on the wheels, but not large enough to be characterized as an overall win (Barton et al., 2017). NMs, on the other hand, are defined as scenarios that players feel as if they are close to winning the game, which are often the symbols on the slot machine reels arranged adjacently in a way that the player almost hit the jackpot, and this effect reinforce the player to be motivated to continue playing the game (Skinner, 1953). For example, the last reel will stop on the symbol or the space between that and the symbol leading to a jackpot, as shown in Figure 1. In addition, we are considering only the

sequential near-miss case, which is the example discussed above, and not the non-sequential near-miss, which the discrepancy in reel stop happens in the second reel. This temporal delivery is essential in reinforcing the motivation of non-addicted individuals to continue gambling (Clark et al., 2009; Worhunsky et al., 2014).

2 Methods

2.1 Model

Using structural and functional connectivity previously explained in literature, we have proposed a dynamic causal model for this near-miss effect which has different implications on the healthy participants and the ones with gambling disorder shown in Figure 2.

In the model, the driving input was chosen to be the sensory input which the individual receives upon a result outcome. Using a slot machine gambling game as the focus of our model, the main sensory input would be visual stimuli. The superior colliculus (SC), located on the dorsal surface of the midbrain, is essential for control of saccadic eye movements and responding to visual stimuli (Basso & May, 2017; Sparks, 2002). Recent studies have also shown that SC is also involved in decision-making (Hasegawa, Hasegawa, & Segraves, 2006) and cognitive circuits (Basso & May, 2017). While other regions of the visual pathway are also likely to be implicated in the receiving and processing of the sensory input during the slot machine gambling game, to simplify the model, SC was used as the sole node for driving input.

Anatomical projections from SC to Substantia Nigra (SN) have been reported (Comoli et al., 2003). Moreover, it has been suggested that SC is involved in mediating activation of dopaminergic neurons by visual stimuli (Redgrave et al., 2010). Dopaminergic neurons located in the SN respond to unexpected biologically salient stimuli, including those associated with reward. Phasic responses of dopamine neurons are critical in signalling the 'reward prediction error', which determines future behavior probabilities through reinforcement learning (Schultz, 2002). Involvement of dopaminergic inputs in gambling is further supported by reports of severe gambling as a side effect of medication used for Parkinson's disease (Dodd et al., 2005).

Gambling severity is suggested as a modulatory input to the SN as Habib et. al have observed that differences in brain activity were found in the left midbrain, near the substantia nigra and ventral tegmental area (SN / VTA), between pathological gamblers and healthy controls (Habib & Dixon, 2010).

The ventral striatum and insula are often implicated in the neural circuits critical for reward processing and incentive-based learning (Pagnoni, Zink, Montague, & Berns, 2002). Abnormal recruitment of these regions has been associated with addiction and risk-taking behaviours (Limbrick-Oldfield et al., 2017). Furthermore, the insula is strongly implicated in causing cognitive distortions during gambling as damage to insula is associated with diminished sensitivity to cognitive distortions (Clark, Studer, Bruss, Tranel, & Bechara, 2014). Connectivity between the insula and the ventral striatum during near-miss outcomes have also been observed to be associated with gambling severity (van Holst, 2014; Chase, & Clark, 2014).

The Superior Colliculus (SC) being node 4 seems to be contradicting the traditional notation of the node receiving driving input as node 1, the reason was that this component was added later to the model and fits perfectly as the extension without needing to modify the code significantly. Then, using MATLAB R2018b (MathWorks), we designed the modulatory and driving inputs, shown in Figure 3 such that each driving inputs are classified as sharp spikes with a magnitude of 5, separated 6 seconds apart, throughout the whole 120-second interval, and the modulatory inputs are separated into three unique blocks, representing the three cases, win, full-miss loss and near-miss loss, respectively.

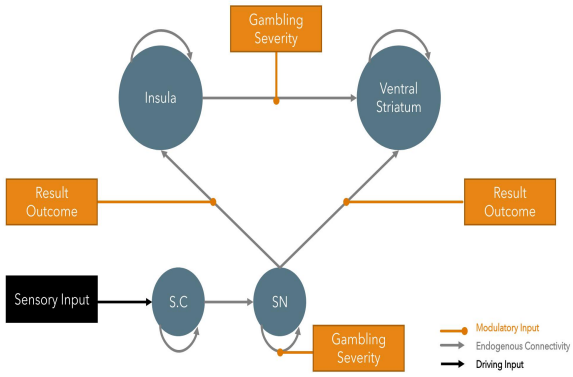

Figure 2: Proposed Model of Causal Connectivity. SC is the first node receiving sensory inputs, then SN receives that projection with modulated self inhibition in gambling addicts case. The signal is then projected to both insula and VStr, also with modulation when encountering different gambling results, win, full-miss loss and near-miss loss. Finally, the signal at insula is then projected to VStr with modulation when encountering near-miss loss scenario in gambling addicts case

2.2 Neuronal Activity

As the proposed model does not have any nonlinear components, modulatory connections from the node to the connection, we have assumed that this is model is a bilinear model. The dynamics of this neural system is implicated by a differential equation in (1), which its component used in this model, A, $B^{a,b}$ and C are matrices shown as (2), (3) and (4) respectively.

$$\frac{dx}{dt} = Ax + u_{a,b}B^{a,b}x + Cu_4 \tag{1}$$

$$A = \begin{bmatrix} -0.1 & 0 & 1 & 0 \\ 1 & -0.1 & 1 & 0 \\ 0 & 0 & -0.1 & 1 \\ 0 & 0 & 0 & -0.1 \end{bmatrix} \tag{2}$$

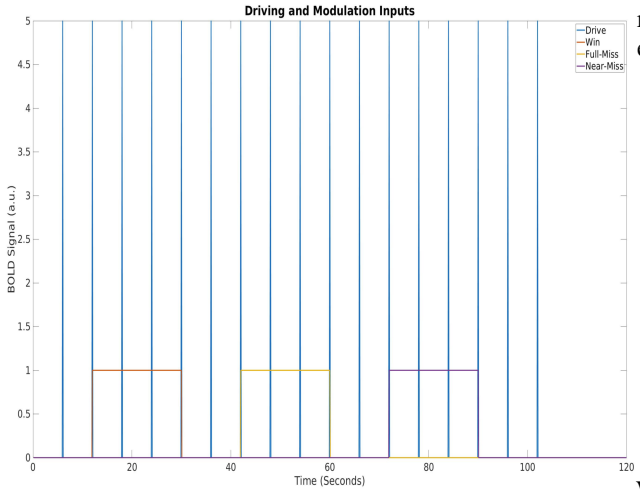

Figure 3: Input Design for the Model

$$B^{a,b} = \begin{bmatrix} 0 & 0 & a & 0 \\ b_{NM} & 0 & a & 0 \\ 0 & 0 & b & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{3}$$

$$C = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} \tag{4}$$

With slight different conditions in cases, of whether the gambling outcomes would be a win, full-miss loss, or a near-miss loss, and whether the individual is a healthy individual or a gambling addict, we have separate cases for comparison in the end by having a represent the gambling outcomes as 1, -1 and 0.3 for win, full-miss loss and near-miss loss, respectively. These values represent the modulatory strengths on the connection between SN and both the insula and VStr, which the near-miss loss, having a value of 0.3, is claimed to be representing the wins, but not as a perfect substitute. In addition, b has a varying value of 0 and -0.5 representing healthy individuals and gambling addicts, respectively. The value of -0.5 signifies the claimed assumption on gambling addicts having a lesser self-inhibition on SN. Finally, the b_{NM} has a value of 0.5 in a unique situation of the gambling addicts going through the near-miss loss scenario, which leads to an increased reinforcing effect on the projection from insula to VStr.

2.3 Blood-Oxygen-Level-Dependent (BOLD) Signal Change

Hemodynamic states of a region, which is associated with how the blood flows in the brain when there is an activity in that region, are a function of neuronal states of that specific region . With that, we are utilizing these four differential equations, which are defining the hemodynamic update.

$$\dot{s} = f_s(x, s, f) = x - \kappa s - \gamma(f - 1) \tag{5}$$

$$\dot{f} = f_f(s) = s \tag{6}$$

$$\dot{v} = f_v(f, v) = \frac{1}{\tau}(f - v^{\frac{1}{\alpha}}) \tag{7}$$

$$\dot{q} = f_q(f, v, q) = \frac{1}{\tau}(f\frac{1-(1-E_0)^{\frac{1}{f}}}{E_0} - v^{\frac{1}{\alpha}}\frac{q}{v}) \tag{8}$$

With κ = 0.64, γ = 0.32, τ = 2, α = 0.32 and E_0 = 0.4, we can then compute these update equations, but not without current hemodynamic state of the region and also its current neural state at time t. After the update calculations, we can then use the value from (8) and (7) to help in calculating the BOLD signal change which are governed by (9) (Friston et al., 2003; Stephan et al., 2007). In addition, since we are assuming that the subject is going through a 3 Tesla fMRI machine, we shall use the predetermined parameters below.

$$\lambda(q, v) = \frac{\Delta S}{S_0} \approx V_0[k_1(1-q) + k_2(1-\frac{q}{v}) + k_3(1-v)] \tag{9}$$

$$k_1 = 4.3\vartheta_0 E_0 TE \tag{9a}$$

$$k_2 = \varepsilon r_0 E_0 TE \tag{9b}$$

$$k_3 = 1 - \varepsilon \tag{9c}$$

V_0 = 0.04 $\quad E_0$ = 0.4 $\quad \vartheta \approx 80.6\ s^{-1}$

$r_0 \approx 110\ s^{-1} \quad TE \approx 0.035\ s \quad \varepsilon \approx 0.47$

With these update equations, we utilized Euler integration technique into updating the neuronal state and BOLD signal change at time t. However, it is important to note that as the hemodynamic parameters are unique for each region, there needs to be a nested loop outside, looping through each region.

3 Results

From Figure 4, the gambling addicts in win situations tend to have an increased neuronal activity in VStr the most with insula, SN and SC coming in second, third and the fourth place, respectively. This increasing trend in neuronal activity projects a monotonic trend on the BOLD signal change in the respective component and also on healthy control's neuronal activity and BOLD signal change, according to Figure 4.

Interestingly, the 'Neural Activity LOSS' plot shows attenuation of the neuronal activity in insula and VStr, in

all gambling severity conditions, going down to the bottom line of zero. The 'Neural Activity NEAR MISS' plot shows increased neuronal activity, at a lesser degree than that of the win situation, in all brain regions except the SC, with an exceptionally higher degree of activity in VStr in the gambling addicts.

The BOLD signal change in insula and VStr for the loss situation of both gambling severity cases do not converge at zero, unlike the neuronal activity of the same situation and gambling severity case. However, the insula BOLD signal change in both cases seem to have reached the minimum at zero and even bounces up from that by an insignificant amount before going up, thus marks the end of loss situation modulation input block. Having a previously higher magnitude, the VStr BOLD signal change in both cases do not seem to have reached the minimum, possibly at zero, yet, since it did not elicit the same bounce up behavior similar to the one seen in insula BOLD signal change, before going back up.

4 Discussion

These observations demonstrate possible causal connections between areas in the brain associated with increased cognitive distortions in pathological gamblers, ones with high gambling severity, compared with healthy controls, ones with low gambling severity. Our plots in Figure 4 show that there is a significant difference between the neuronal activity in VStr of the gambling addicts and that of the healthy controls in win and near-miss loss cases, which is the specific scenario found to be affecting the VStr more in gambling addicts only, so the modulation effects were additionally compounded along with the original gambling outcome modulation effects (van Holst, Chase, & Clark, 2014).

These aspects of the model which fit with the literature are essential for strengthening our hypotheses that there are connections between brain areas processing these results in a way that an effective diagnosis on gambling disorders can be made from measuring these brain areas' activity when going through the gambling task, and also to understand the cognitive distortions in these brain areas. Furthermore, using fMRI, which uses BOLD signal contrast, one could utilize the proposed model in differentiating between win and near-miss scenario through insula BOLD signal contrast, which the difference between the signal of gambling addicts and that of healthy controls are far more significant than those of VStr, which can be seen in Figure 4 .

However, there are several notable limitations and issues that were not thought of well enough. First, even though SC was chosen as the most probable candidate for receiving and relaying inputs to SN due to its direct connection between these two brain areas, we have left out the fact that the main purpose of SC is to identify motion of the objects (Davidson and Bender, 1991), and not the object identification, which is what the gambling participants in real world scenario are doing when interacting with the gambling. In addition, the brain area that processes auditory information, auditory cortex, was even left out of this model even though the audio cues in gambling environment do have an effect on gamblers' time used for reflecting and thinking before acting after losses (Brevers et al., 2014). Second, there is an issue of practicality in implementation of the model to real world application, which focuses on how the noise, whether from the lack of more optimized instruments or limitations of the instrument itself, affect the signal measurements, which consequently, affect the interpretation of gambling severity. In this case, we are referring to taking measurements from SN, SC, VStr and insula, which might not be trivial since there might be confounders, such as Thalamus which connects to both Nucleus Accumbens, part of VStr, and insula (Cho et al., 2013). Furthermore, there is a paper contradicting the hypothesis of increased response to near-miss outcomes in pathological gamblers, in addition to discussing that near-miss and loss outcomes may be less salient in pathological gamblers, deriving from the issue of them having blunted loss responses (Worhunsky et al., 2014). In short, SC mainly detects motion of the object, not identifying objects, making it not an ideal candidate despite the direct connection to SN, and these areas' noises in measurements via fMRI might make this model implausible in real world application, not to mention the contradictory findings in literature which needed further exploration on the issue.

These issues and limitations to our model give rise to questions implicating future directions for pushing this project forward, making it more applicable to real world applications of detecting and ideally, predicting gambling severity of an individual. First, we can extend the model to cover more areas of the brain and connect it to other relevant physiological responses. For example, as it is known that insula outputs to thalamus and motor cortex, which thalamus, being known as a relay station to the somatosensory system, can be an intermediary node connecting between insula and somatosensory system (Allen et al., 2016). The component of somatosensory system that is being focused on is the skin conductance response (SCRs), which is found to be correlated with how an individual processes rewards in gambling games (Lole, Gonsalvez, Barry, & Blaszczynski, 2014). In addition, a pattern of large SCRs is presented in near-misses situations with jackpot symbols landing on the first two reels, compared with other types of near-misses and regular losses (Dixon et al., 2012). Thus, it is interesting to explore the possibility of extending the model by measuring skin conductance responses simultaneously ,while the individual is going through experimental slot-machine fMRI task used in (Worhunsky et al., 2014), but ideally with an extension of recruiting more female participants to make the number of participants from each gender equal to each other.

Consequently, with experimental data that can be used as evidence, we can now, based on the evidence, fine tune the model weights and perform model comparison between our proposed model and other models that we also hypothesized based on the literature, using negative free energy.

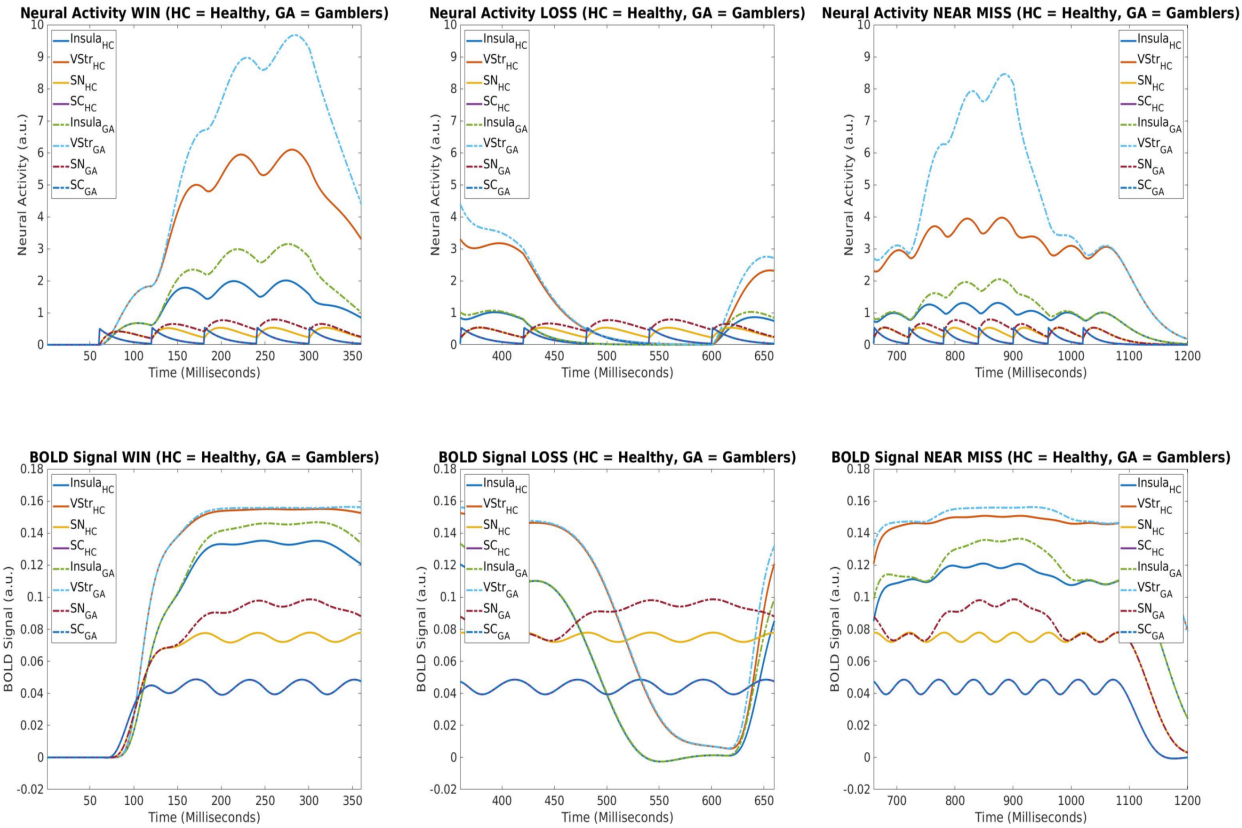

Figure 4: Neuronal Activity and BOLD Signal Change Outputs

Ideally, the model could predict an individual performance on Gambling Disorder Screening Questionnaire (GDSQ), a self-report questionnaire based on the DSM-IV and DSM-5 criteria for Gambling Disorder (Villella et al., 2016), from having the patient do the task, which would strengthen the hypothesis. Then, for visualization and education purposes, we can deploy the graphical user interface of a simulation integrated with the best model, outputting a real time feed to simulate the relationship and effect of each scenario on each and every level of gambling severity.

Second, as a practical application for alleviating the gambling severity, we suggest on making an application, either a web application or a phone application, with interactive games or tasks that can infer upon the gambling severity by using algorithm to process on collected information, electrodermal activity from possibly a device measuring skin conductance response that can contact with the application, self assessment, GDSQ or questionnaire asking on their motivation to continue playing, and latency of response. With those data, and results, there should also be additional validation using existing diagnostic criteria on DSM-V and medical experts' opinions, all of which are crucial in this stage. Then, to alleviate the effect of gambling, we shall utilize the method used with quitting smoke, by decreasing the amount of the stimulus gradually, meaning that, given a progress to the player, there will be games that involve risk and uncertainty in a way that it mimics the mechanism of gambling machines, and as the player, assuming that they are addicted to the game, progress further, there will be some kind of additional game rules introduced to the player in a way that will reduce the time spent on it, and gradually increase the rules, which in turn decrease the amount of games played. Ideally, the player will eliminate this gambling disorder. However, future work is needed to determine the rules and mechanisms of the game which will mechanistically help the player eliminate this gambling addiction or gambling disorder.

In conclusion, despite limitations of the measurement noises making the model implausible to be applied with real world application, and utilizing the brain area that deem irrelevant in image recognition, the proposed model did provide a possibility of causal connections between areas in the brains of gambling addicts and healthy controls, which showed cognitive distortions, defined as gambling severity, to some degree. Furthermore, there is a need to explore the issue of extending the model into somatosensory systems, which in this case is utilizing skin conductance responses, and ideally, use all aspects of the model to predict a patient's performance on GDSQ.

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